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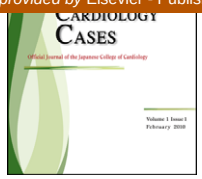

Case report

Characteristics of *in vivo* images from an in-stent restenosis lesion of a saphenous vein graft after bare-metal stent implantation: Assessment using optical coherence tomography

Daiji Yoshikawa (MD)[a,*], Hideki Ishii (MD, PhD)[a], Yutaka Aoyama (MD)[a], Kyoko Matsudaira (MD)[a], Takuya Okada (MD)[a], Yasuhiro Uchida (MD)[a], Hitoshi Ichimiya (MD)[a], Yuuki Shimizu (MD)[a], Satoshi Isobe (MD, PhD)[b], Toyoaki Murohara (MD, PhD)[a]

[a] *Department of Cardiology, Nagoya University Graduate School of Medicine, 65 Tsurumai-cho, Shouwa-ku, Nagoya 466-8550, Japan*

[b] *Department of Cardiology, Kami-iida Daiichi General Hospital, Nagoya, Japan*

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Summary Restenosis of saphenous vein grafts (SVG) after bare-metal stent (BMS) implantation remains a clinical problem. Recently, intravascular optical coherence tomography (OCT) has been proposed as a high resolution intravascular imaging modality, and is able to distinguish several components of intracoronary structures. *In vivo* images of in-stent restenosis (ISR) lesions in an SVG using OCT have not been reported. In this case report, we present the characteristics of *in vivo* OCT images from an ISR lesion of an SVG after BMS implantation.

Case report

A 72-year-old female with obesity (body mass index = 26.4 kg/m^2), hypertension, hyperlipidemia, diabetes mellitus, and chronic kidney disease (estimated glomerular filtration rate = 36.4 mg/m^2/1.73) was admitted to our facility due to recurrent chest pain. At 52 years of age, this patient underwent coronary bypass surgery [saphenous vein graft (SVG) to the left anterior descending and left internal thoracic artery to diagonal branch] to treat angina pectoris. At 68 years of age, a bare-metal stent (BMS) (Driver®, 3.5 × 15 mm, Medtronic Vascular, Santa Rosa, CA, USA) was implanted at a *de novo* stenosis site in the SVG to resolve unstable angina. After the stent implantation, her hyperlipidemia was well treated with statin, however, diabetes mellitus was poorly controlled in spite of insulin

* Corresponding author. Tel.: +81 52 741 2147; fax: +81 52 744 2138.
E-mail address: yodaiji_0526@yahoo.co.jp (D. Yoshikawa).

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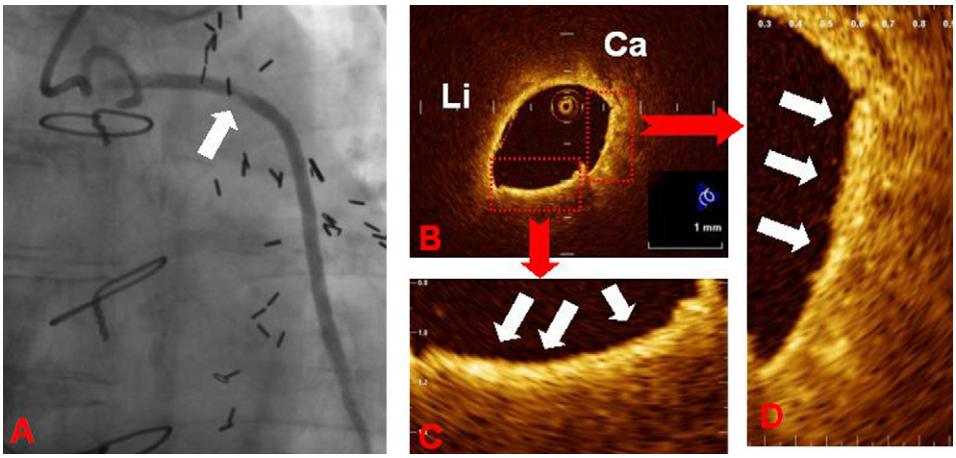

Figure 1 (A) and (B) The culprit lesion shows a lipid-rich plaque with some calcification (Li: lipid-rich plaque, Ca: calcified plaque). (C) and (D) Plaque composition (from 2 to 8 o'clock) appears to be composed of a layer of fibrotic tissue with a high intensity spotty area with attenuation or acoustic shadowing suggesting an accumulation of macrophages (white arrows).

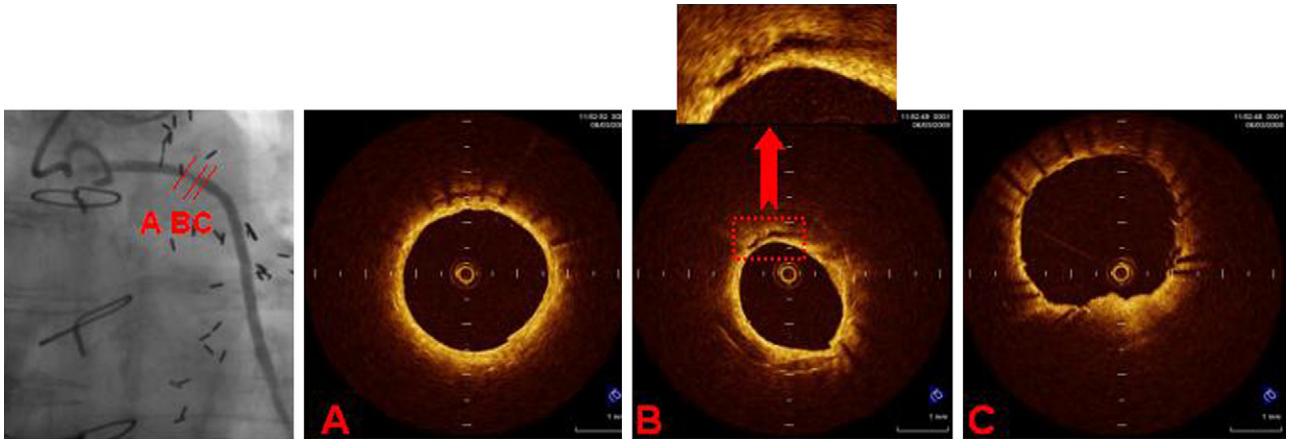

Figure 2 (A) At the proximal culprit lesion, eccentric circumferential fibrous tissue is observed covering all stent struts. (B) At the distal culprit lesion, the prevalence of calcified plaque between stent struts and the lumen is clearly seen, and a small microchannel (red arrow) is observed at 11 o'clock. (C) Low attenuation and high signal intensity indicate white thrombus attached to the stent surface. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of the article.)

use (hemoglobin A1c: 7.5—8.0%). Four years later, a coronary angiogram revealed a severe in-stent restenosis (ISR) lesion of the SVG, as shown by optical coherence tomography (OCT) before coronary intervention (Fig. 1, Panel A). The culprit lesion (minimum lumen area = 1.70 mm^2) showed lipid-rich plaque with partial calcification (Fig. 1, Panel B). Plaque content (from 2 to 4 o'clock and again from 4 to 7 o'clock) was composed of a layer of fibrotic tissue with a high intensity spotty area with attenuation or acoustic shadowing suggesting an accumulation of macrophages [1] (Fig. 1, Panels C and D). Eccentric, circumferential, fibrous tissue was observed covering all stent struts at the proximal culprit lesion (Fig. 2, Panel A). At the distal culprit lesion, there was clear prevalence of calcified plaques between stent struts and the lumen, and a small microchannel was observed at the direction of 11 o'clock (Fig. 2, Panel B). Low attenuation and high signal intensity, indicating possible white thrombus, was observed attached to the stent surface (Fig. 2, Panel C). After balloon inflation, a drug-eluting stent was implanted at the culprit lesion.

Discussion

In native coronary arteries, ISR after BMS implantation is caused by neointimal proliferation consisting of smooth muscle cells, and is revealed as a homogeneous, signal-rich structure by OCT [2]. On the other hand, ISR of SVG after BMS implantation is primarily due to atherosclerotic plaque or fibromuscular hyperplasia, and secondarily due to thrombus formation [3]. However, until now, limited information has been available regarding the characteristics of *in vivo* OCT images of SVG after coronary stent implantation. For the case presented, the ISR lesion of the SVG was evaluated by OCT, which has recently been proposed as a high

resolution intravascular imaging method (10 μm∼) [4]. Our OCT findings showed that the ISR lesion in SVG composed of lipid-rich and calcified plaques, fibrous hyperplasia, and intramural thrombus.

For this case, we speculate the mechanisms for significant restenosis at the stent site are as follows: in addition to chronic endothelial cell injury and dysfunction of the degenerated SVG, inflammation caused by stent implantation promoted not only neointimal tissue proliferation but also atherosclerosis progression, as well as other potential causes. Stent thrombosis might result from severe upstream stenosis causing reduction in flow. A previous autopsy study demonstrated that newly formed vessels are seen in the atherosclerotic intima of the native coronary artery [5]. In this case, neovascularization may be indicated by the microchannel that occurred in the ISR lesion of SVG.

The strategy of percutaneous coronary intervention (PCI) for ISR lesions of SVG is not well established due to the unpredictability of distal embolization at the time of PCI [6]. Detailed intracoronary imaging may provide more important clarifying information regarding this issue. In this case report, to the best of our knowledge, we present for the first time the characteristics of *in vivo* OCT images obtained from an ISR lesion of an SVG after BMS implantation.

References

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